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Review

A review of the mechanisms and modeling of photocatalytic disinfection

Omatoyo K. Dalrymple[a,*], Elias Stefanakos[a], Maya A. Trotz[b], D. Yogi Goswami[a]

[a] Clean Energy Research Center, University of South Florida, Tampa, FL 33620, United States
[b] Department of Civil & Environmental Engineering, University of South Florida, Tampa, FL 33620, United States

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ABSTRACT

This paper is a review of the fundamental disinfection mechanisms of photocatalysis and the models used to fit the disinfection process. Photocatalysis is an attractive technology for water treatment largely due to its potential to utilize solar energy directly and achieve both disinfection and chemical detoxification. Many papers have been published on photocatalytic disinfection, but there is still considerable debate on disinfection mechanisms and a general lack of mechanistic models for the process. The fundamental photocatalytic disinfection mechanisms as they relate to the inactivation of bacteria are comprehensively surveyed here. The process of lipid peroxidation of membrane fatty acids, particularly polyunsaturated fatty acids, is gaining momentum in the literature. In recent papers, an increasing number of researchers are paying close attention to the products of lipid peroxidation.

The mathematical models, empirical and mechanistic, used to fit the disinfection process have also been thoroughly reviewed. In this regard, most of the proposed models are empirical in nature and rooted in traditional chemical disinfection principles, which are often not representative of the heterogeneous photocatalytic process. The theoretical development of a mechanistic model for photocatalytic disinfection based on lipid peroxidation is explored with due consideration to the interaction between microbes and photocatalyst particles. The extensive literature on autooxidation of lipids in such fields as biology and medicine is informative to the development of the model.

Contents

* Corresponding author. Tel.: +1 813 974 6614; fax: +1 813 974 6438.
E-mail address: odalrymp@mail.usf.edu (O.K. Dalrymple).

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1. Introduction

Waterborne diseases have been transmitted through microorganisms such as viruses, bacteria and protozoa. To ensure the safety of consumers, drinking water is disinfected and this has traditionally been achieved by the addition of chlorine. However, concerns about the formation of potentially mutagenic and carcinogenic disinfection byproducts (DBP) during water chlorination have led to the development of many alternative processes for the disinfection of drinking water [1,2]. The control of DBPs has become important. These compounds are formed from the reaction of chlorine with natural organics in water and include trihalomethanes (THMs) and haloacetic acids (HAAs). Recent Environmental Protection Agency (EPA) regulations have further limited THMs, HAAs and other DBPs (including chlorite and bromate) in drinking water [3]. As a result, many water systems now limit the use of chlorine to high-quality groundwater or reduce total organic carbon prior to disinfection.

Another concern of traditional disinfection is that some organisms tend to develop resistance to chlorine or require higher than normal doses for complete inactivation [4,5]. Relatively high residual chlorine concentration can make drinking water taste and smell unpleasant. Nonetheless, chlorination remains an important disinfection method.

Alternative disinfection processes include ozonation, use of chlorine dioxide, advanced filtration processes, and germicidal ultraviolet (UV) radiation among others. However, even though they can be effective, many of these alternative processes require expensive chemicals or costly equipment to generate the disinfectant onsite. This is particularly true for ozone and chlorine dioxide. In general, they are often associated with increased costs and process complexity. UV disinfection makes use of shortwave radiation (<280 nm) which requires the set up of expensive lighting equipment and is associated with increased energy utilization. Moreover, ozonation has also been shown to result in potentially harmful byproducts including bromate and other brominated DBPs formed in waters with elevated bromide [6,7].

Among the alternatives, photocatalysis is becoming a viable option because of its potential to use sunlight to drive the disinfection process using a solid catalyst such as titanium dioxide (TiO_2). The highly reactive hydroxyl radical ($^{\bullet}OH$) serves as the main oxidant and is capable of inactivating microorganisms, including viruses, bacteria, spores and protozoa.

Photocatalysis was first shown to be an effective sterilization process by Matsunaga et al. [8], who reported on the killing of *L. acidophilus, S. cerevisiae* and *E. coli*. Many other researchers have since reported on the use of photocatalysis for water disinfection. By far the most studied organism in the literature is *E. coli*, largely used as an indicator organism in drinking water systems; see for example [9–19]. Cheng et al. [20] reported on the inactivation of several strains of *L. pneumophila*. The inactivation of *Cryptosporidium* and *Giardia*, known for their resistance to many chemical disinfectants, including chlorine, was reported by a number of researchers [21–25].

Even though previous researchers had considered solar photocatalysis for oxidation of chemicals, Block et al. [26] were among the first researchers to explore the use of solar illumination to drive the disinfection process. The engineering and economical feasibility of these systems were explored in detailed by Goswami [27] and Goswami et al. [28]. Although they are not currently in widespread use, solar photocatalytic systems have been used with much success in pilot facilities [29–31]. These systems are particularly adaptable in developing countries. They offer huge energy savings and are particularly adaptable for applications in remote and rural areas, where energy supply may be prohibitive [32]. In addition, TiO_2 is abundant and relatively cheap.

However, photocatalytic treatment has its challenges. With TiO_2 as the photocatalyst, only the small UV portion of sunlight (<5%) is utilized for the process. Catalyst modification techniques have been attempted to shift its light absorption capacity towards visible wavelengths [33,34]. Most of these techniques result in lower overall photocatalytic activity, even if there is some increase in activity in the visible spectrum [35–38]. Also, the process is much more efficient when the catalyst is present as a micro- or nano-particle in suspension. The risk of nano-particle contamination and the need to regenerate the catalyst suggest that post-treatment separation is required, adding a level of complexity and increased cost. Nonetheless, this challenge is solved by using reactors in which the catalyst has been immobilized on an appropriate surface such as glass [12,19,39,40], membranes [12,41], polymers [42–45], metal [46], and fibers [46–49]. Hall et al. [50] and Beydoun et al. [51] attempted the use of magnetism to help retain or recover the catalyst by attachment to magnetic particles. Even though some of these applications have lower disinfection efficiency than slurry reactors, they improve the practicality of the technology overall. For example, the coating of TiO_2 to surfaces has helped to expand disinfection functionality to medical implants [52].

One of the biggest challenges is the lack of reliable models to describe the inactivation process so that the long-term goal of optimizing photocatalytic disinfection can be achieved. The mechanism of inactivation for common indicator organisms such as *E. coli* is still heavily debated. Most of the previous analyses have been based on empirical models, but there is no reason to believe that these models can be extrapolated beyond the range of values for which they have been calibrated. Further, the available models do not adequately and consistently describe photocatalytic disinfection, largely because they are rooted in conventional disinfection which is based on the chemistry of homogeneous reactions.

In order to improve its potential for water disinfection, design methodologies must be developed for photocatalytic disinfection systems. Once these methodologies are developed, they can be optimized so that water may be disinfected quickly, efficiently and inexpensively. The information needed for the design includes knowledge of the rate of inactivation of the target or indicator organism(s). In particular, the effect of catalyst dose and light intensity on the rate of the process will determine the most efficient combination of contact time, catalyst dose and light intensity to employ.

There has also been relatively little work done in the area of byproduct identification and analysis for photocatalytic disinfection. This is important because disinfection is conducted in waters containing natural organic matter and other contaminants. In the few studies conducted the results vary depending on the contam-

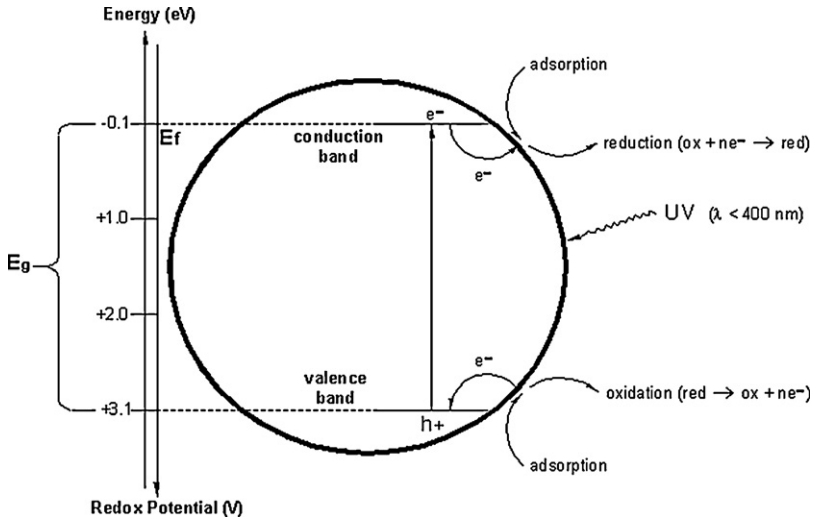

Fig. 1. Scheme of photocatalytic process over TiO_2 surface.

inant of interest. In some studies the toxicity and recalcitrance of the byproducts were higher than that of the parent contaminant [53]. Yet in other work, photocatalysis was effective at reducing the toxicity of the primary contaminant including the endotoxins of microorganisms [16,54,55].

A number of excellent reviews exist on the subject of photocatalysis [56,57], including photocatalytic detoxification and disinfection [15,31,58–60]. Of these, the most recent comprehensive review was conducted by Malato et al. [60]. The goal here is not to replicate the work of these authors, but specifically, to review in detail the various models which have been proposed, the mechanism of inactivation suggested by researchers, and the approach taken to design systems, including the popular concentration–time (*CT*) approach used for chlorine disinfection. The potential benefits and shortcomings of these various proposals are considered. The development of mechanistic models for photocatalytic disinfection is also explored in theory.

2. Background

During the process of photocatalysis a solid semiconductor catalyst generates reactive oxygen species (ROS) on its surface when exposed to light of the appropriate wavelength. These species in turn reduce and oxidize compounds which are adsorbed on the catalyst surface. TiO_2 has become the preferred photocatalyst for a variety of reasons, which include its low cost, chemical stability, non-toxicity, and effectiveness under near-ultraviolet light (300–400 nm). By definition, the catalyst participates and accelerates the transformation of compounds, itself remaining unaltered at the end of each catalytic cycle. The process has been well studied for the decomposition of organic substrates [57,61,62].

The photocatalyst is an inorganic semiconductor material whose energy level structure is well described by quantum theory of solids. These materials include TiO_2, WO_3, WS_2, CdS, ZnO_2, Fe_2O_3 and ZnS. Electron–hole pairs are generated when the material absorbs the energy of impinging photons having equivalent or excess energy ($h\nu$) to the band gap. This means that an electron in the valance band gains sufficient energy to overcome the band gap and reaches the conduction band, with the concomitant vacancy in the valance band (the hole). The band gap, in semiconductor theory, is the void energy region which separates the valence band from the conduction band (see Fig. 1). For TiO_2, the band gap can be overcome with energy from near UV photons. The absorption of energy and the subsequent generation of the electron–hole pair are the initiating steps and may be represented as follows:

$$TiO_2 + h\nu \rightarrow e^-_{cb}(TiO_2) + h^+_{vb}(TiO_2) \quad (1)$$

where e^-_{cb} is the conduction band electron and h^+_{vb} is the valence band hole.

The interaction of the hole with water molecules or hydroxide ions produces the very reactive hydroxyl radicals. These radicals are bound to the surface of the hydrated metal oxide and act as the primary oxidants in the photocatalytic system [63,64]. The formation of the radicals is illustrated below.

$$Ti^{IV}|H_2O + h^+_{vb} \rightarrow Ti^{IV}|\bullet OH + H^+ \quad (2)$$

$$Ti^{IV}|OH^- + h^+_{vb} \rightarrow Ti^{IV}|\bullet OH \quad (3)$$

Oxidation of compounds may also occur directly via the valence band hole before it is trapped either within the particle or at the particle's surface. Nevertheless, the presence of hydroxyl radicals in aqueous solutions of illuminated TiO_2 has been confirmed by researchers, and many intermediates are consistent with those found when organic compounds react with a known source of hydroxyl radicals [65–68]. The chemical properties of the compound and the reaction conditions largely determine which mechanism will dominate. However, the presence of hydroxyl radicals is very important for the complete photocatalytic destruction of many organic compounds and the inactivation of pathogens. Cho et al. [69] found a linear correlation between hydroxyl radicals and the inactivation of *E. coli* in water disinfection studies.

The photo-generated conduction band electrons are trapped at the surface by Ti^{IV} sites and result in Ti^{III} sites. Oxygen adsorbed at Ti^{III} sites may result in ROS such as the superoxide radical from a charge transfer reaction:

$$Ti^{IV} + e^-_{cb} \leftrightarrow Ti^{III} \quad (4)$$

$$Ti^{III} + O_2 \leftrightarrow Ti^{IV} + O_2^{\bullet -} \quad (5)$$

It is not surprising that photocatalysis, which has proven effective in degrading many organic and inorganic compounds, would also exhibit biocidal properties. After all, microorganisms, such as bacterial cells, are 70–90% water and the major cellular constituents, such as polysaccharides, lipids, proteins and nucleic acids, can be attacked by ROS and subsequently lead to cell death. Matsunaga et al. [8] were the first to demonstrate the biocidal activity of TiO_2. Since then, there has been great interest in using the process for a variety of applications in relation to disinfection. The ability to use solar light to drive the process remains particularly attractive

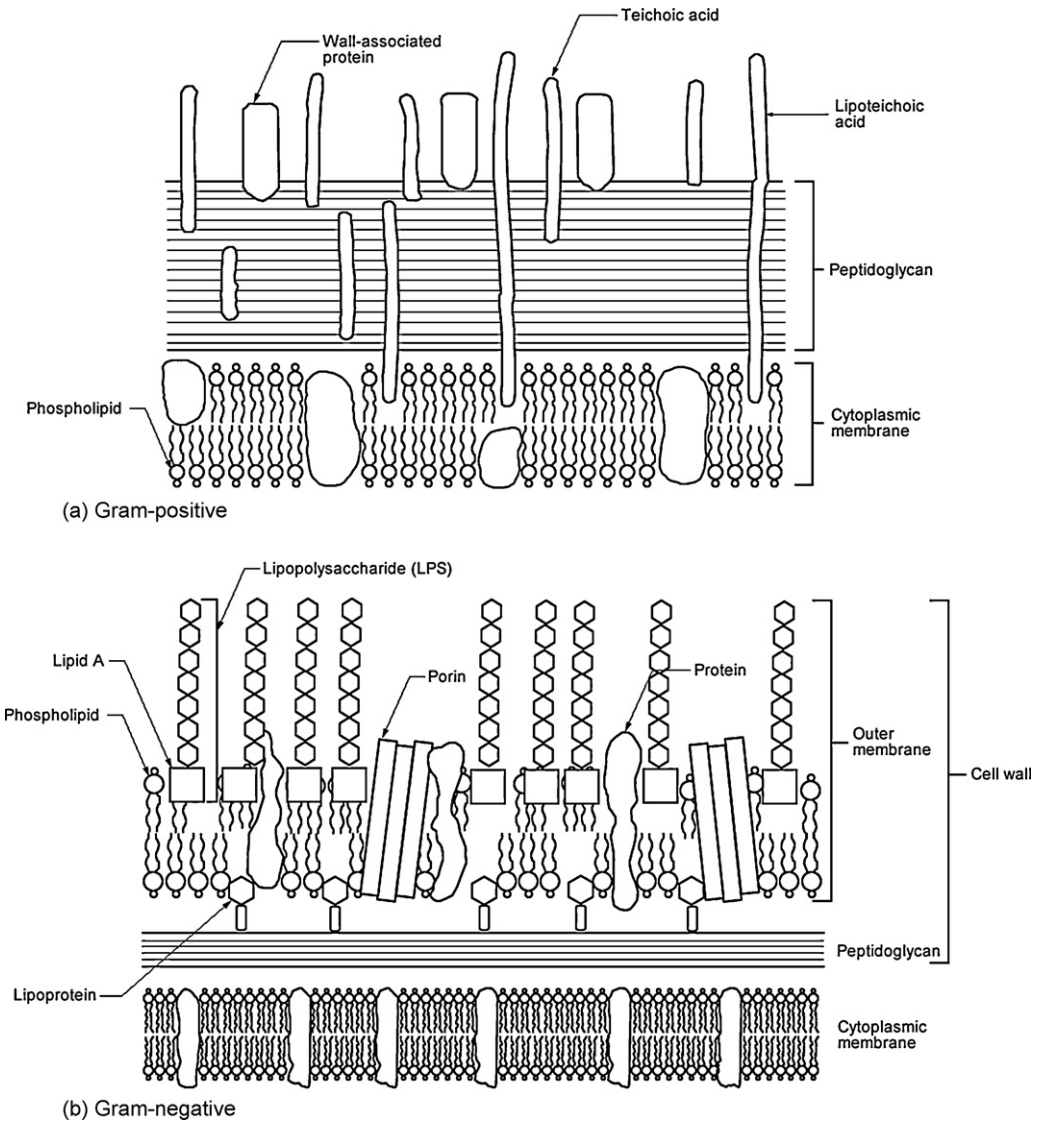

Fig. 2. Outer layers of bacteria: (a) Gram-positive (b) Gram-negative.

and a few pilot plants have shown that disinfection is achievable under solar conditions [11,23,25].

3. Photocatalytic disinfection mechanisms

Defining the mechanism which results in microbial inactivation has been the preoccupation of many researchers. There is still much debate over which process or set of processes lead to death of an organism exposed to photocatalytic action. However, most of the research now indicates that the destruction of the cell membrane is an important process for inactivation.

It has been known for a while that, if given sufficient time, photocatalysis would eventually oxidize most of the organic material which constitutes microorganisms [70]. As much as 96% of the cell's dry weight is composed of organic macromolecules, which include proteins, polysaccharides, lipids, lipopolysaccharide and nucleic acids (DNA and RNA). Monomers are 3% of the dry weight and include amino acids, sugars, nucleotides and monomer precursors. The remaining 1% is inorganic ions [71]. It has been successfully shown that TiO_2 can inflict severe damage on molecules such as amino acids [72,73] and DNA [74] when treated in isolation.

However, many of these molecules are complex, and collectively, possess different means of chemical and physical resilience to ensure the survival of the cell. There are, for example, several classes of phospholipids, each of which exists in many kinds due to variability in fatty acid composition among species based on genetic differences and growth conditions [75,76]. Not only are the individual properties of these constituents important, but also the manner in which they are structurally arranged (for example, in lipid bilayers or cross-linked in peptide chains) confers additional resistance to inactivation.

Of principal concern to researchers, however, are:

(1) Which specific set of processes lead to cell inactivation (inability to replicate and eventual death)?
(2) What is the length of time for cell inactivation to occur?
(3) How do these processes differ among species of microorganisms?

A number of different mechanisms have been suggested. Rather than presenting these mechanisms in isolation, the following discussion is based on a systematic deconstruction of the cell structure, highlighting the most important targets identified as playing sig-

nificant roles in the overall inactivation process. For simplicity, the various sites targeted during photocatalytic inactivation are grouped as extracellular and intracellular.

3.1. Extracellular target sites

The cell membrane and cell wall are obvious targets in the extracellular environment. However, these outer layers are themselves complex structures with multiple sites for attack. In the case of bacteria, at least three classifications can be established: (1) the peptidoglycan layer, present in both Gram-negative and Gram-positive bacteria; (2) the lipopolysaccharide layer (LPS), the outermost layer found only in Gram-negative bacteria; and (3) the phospholipid bilayer—two in Gram-negative and one in Gram-positive organisms.

3.1.1. Peptidoglycan

Not much has been published on the effects of photocatalytic attack of the peptidoglycan layer. Peptidoglycan is a peptide-cross-linked polysaccharide matrix that surrounds the cell (Fig. 2). The basic structure is a sheet formed from individual strands of peptidoglycan lying adjacent to one another. In Gram-positive bacteria, this layer can account for as much as 90% of the cell wall with several (up to 25) sheets stacked upon each other. In Gram-negative bacteria, it makes up only about 10% of the cell wall. Peptidoglycan confers rigidity to maintain shape and internal pressure.

It is likely that the peptidoglycan layer may be susceptible to radical attack [77]. However, in both Gram-negative and Gram-positive bacteria, peptidoglycan is very porous and allows particles of approximately 2 nm to pass through [78]. While these pores are sufficient to allow the passage of oxidative species, such as the hydroxyl radical and superoxide, it may still be difficult for such molecules to permeate to the inner membrane due to their reactivity in the external environment. If inactivation were a function of peptidoglycan thickness, Gram-positive bacteria would be expected to gain the advantage in surviving photocatalytic attack. This has been suggested in the literature by researchers who have exposed species of the two groups to the same photocatalytic conditions [79,80].

However, the comparison between inactivation rates of Gram-positive and Gram-negative species based simply on peptidoglycan layer thickness is of little value due to the difference in location of the layers in each group. In fact, the comparison is based on the complexity and density of the cell wall as a whole. More specifically, it is the ability of the thick peptidoglycan layer in Gram-positive bacteria to impart greater resistance than the outer membrane of the Gram-negative bacteria, given that each represents the first line of defense.

Nonetheless, it has not been established whether the peptidoglycan layer is an actual critical target of attack by radicals or functions to simply retard the diffusion of oxidants to the underlying vital sites, particularly in Gram-positive bacteria. While some researchers did observe a decrease in inactivation rate with increased layer density and complexity [80], it is still difficult to make definitive conclusions about the role of peptidoglycan in the mechanism of inactivation (or in the resistance thereof), since cell wall complexity is a nebulous parameter. It is not known whether consideration was given in these studies to other subtle differences among the organisms, such as membrane lipid content and composition.

3.1.2. Lipids and polysaccharides

To date, the most convincing research has indicated that lipids, particularly polyunsaturated fatty acids (PUFA), are the major targets for oxidative radical attack. The inhibiting effects of lipid peroxidation in cells have been demonstrated both in photocatalytic experiments [9,81] and other reactions involving radicals [82–84]. Polyunsaturated fatty acids are among the most oxygen sensitive molecules in nature.

The effect of ROS, especially free radicals, on cellular molecules has long been reported—see for example [85,86]. These radicals can naturally occur in biological systems, and when not controlled, are responsible for damage to important cell components such as nucleic acids, proteins, and lipids. In humans, lipid peroxidation has been linked to degenerative diseases of aging such as cancer, cardiovascular disease, immune system decline and Alzheimer's disease [87].

In photocatalytic disinfection, lipid peroxidation is of interest because the LPS layer and the phospholipid bilayer are made up of fatty acids, which may be susceptible to peroxidation. Hydroxyl radicals generated in the extracellular environment are likely to travel only very short distances before encountering oxidizable substrate such as fatty acids. The presence of an intact membrane reduces the probability of the radical reaching intracellular components such as DNA. Once the radicals are generated in close proximity to the target molecules, they will be able to inflict injury directly.

In general, the predominant fatty acid in lipid A of *E. coli* is tetradecanoic acid (C14:0), but monounsaturated fatty acids, such as hexadecenoic (C16:1) and octadecenoic (C18:1) acids, may be present in other species [88]. Apart from contributing to structural integrity, LPS is of crucial importance to Gram-negative bacteria, which can be inactivated if it is altered or removed. LPS is associated with endotoxin activity, particularly the lipid A component. The polysaccharide components are associated with immunogenicity.

Even though the exact mechanism has not been elucidated, the degradation of lipid A (and by extension LPS) in *E. coli* has been demonstrated [16]. The destruction of LPS is important not only as a factor in overall inactivation, but also in neutralizing the toxic effects which may still exist after an organism is incapacitated.

A more abundant source of lipids is the phospholipid bilayer membranes. Gram-negative bacteria have two distinct membranes as illustrated in Fig. 2, while Gram-positive bacteria have only one such membrane beneath the thick peptidoglycan layer. These phospholipid membranes are composed of conventional glycerol-phospholipids, mainly phosphatidylethanolamine, phosphatidylglycerol, and cardiolipin [75,76,89].

Because the phospholipid membranes are predominantly composed of a repeating arrangement of lipids, a radical chain reaction initiated by ROS may allow cell injury to occur at sites relatively distant from the initiation source. This occurs because the reaction of an unsaturated fatty acid with a radical in the presence of oxygen leads to the formation of a peroxyl radical, which in turn can react with other nearby lipid molecules to generate additional lipid radicals [85,86]. The process is propagated as these newly formed lipid radicals react with other unsaturated lipids (Fig. 3). The chain reaction eventually results in the oxidation of biomolecules at sites considerably distant from where the initial free radical reaction occurred [90].

The oxidation of membrane lipids was first confirmed by Maness et al. [9] and then later by other researchers [20,77,81,91]. In the former case, the production of malondialdehyde (MDA), the most widely used biomarker for lipid peroxidation, was assessed. They found a steady increase in MDA associated with cell inactivation [9]. The eventual impacts of membrane peroxidation are changes in cell permeability and disruption of the intact membrane, both leading to the release of cell cytoplasm [20,92] and inhibition of membrane-mediated respiration [9].

Though its occurrence has been validated, it is still very unclear how the rate of lipid peroxidation is related to overall inactivation rate and whether a model can be based on this relationship. It becomes obvious that the fatty acid content and composition

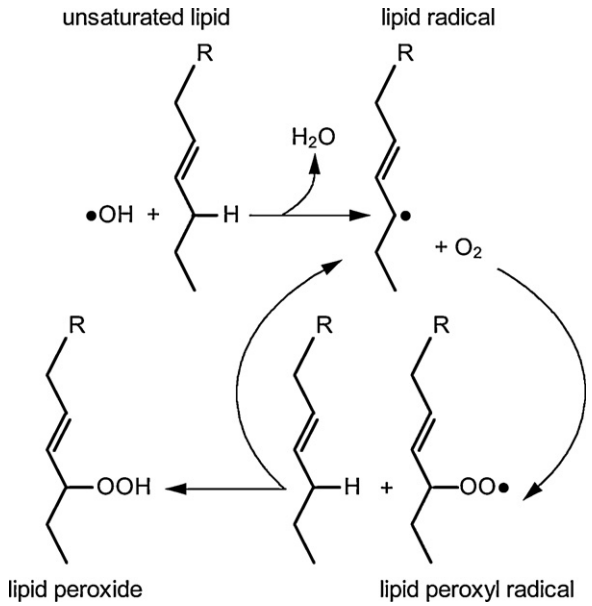

Fig. 3. Schematic of lipid peroxidation.

of cells are critical parameters to estimate the rate of peroxidation, particularly the reactive lipid content. It is now becoming apparent that low reactive lipid concentration in organisms may increase their resistance to photocatalysis as demonstrated by Cheng et al. [20]. They analyzed fatty acids of different strains of *Legionella pneumophila* and were able to infer that fatty acid composition of membrane lipids (saturated versus unsaturated) is among the determining factors for susceptibility to photocatalysis.

These findings raise at least two interesting questions: (1) can the rate of peroxidation be predicted based on the fatty acid content and composition of cells? (2) Can the rate of peroxidation be related to the overall rate of inactivation? The answers to these questions will provide the basis for incorporating lipid peroxidation into a mechanistic model for photocatalytic disinfection, at least for bacteria.

3.2. Intracellular target sites

The complex mixture of substances and structures in the cell is collectively called the cytoplasm. In the cytoplasm, there exist DNA, RNA and ribosomes along with other dissolved or suspended materials. All these substances are important for the proper functioning of the organism. Of course, the cytoplasm is protected by the cell membrane, which is impermeable to most essential molecules and ions. Only water and a few other small, uncharged molecules like oxygen and carbon dioxide diffuse freely across the membrane. All other substances enter through active transport or diffuse through trans-membrane proteins, whose channels open and close according to the needs of the cell.

Three major factors limit the possibility of intracellular target sites being reached: (1) under typical water treatment conditions (pH 5–8), photocatalyst particles aggregate to form composites greater than 300 nm [93,94]; (2) it is usually assumed that oxidation takes place through surface-bound radicals which are not free to diffuse into the cell [8,95]; (3) even if some radicals become detached in solution, they would be highly reactive and likely to encounter oxidizable substrate on the cytoplasmic membrane. This means that attack of intracellular constituents must take place through the generation of other oxidants, such as lipid radicals, hydrogen peroxide and superoxide, or surface-bound radicals on particles which breach the membrane as a result of large perforations in the membrane [20,77,96].

Apart from direct attack, superoxide and hydrogen peroxide can produce hydroxyl radicals in the intracellular environment through the Fenton reaction involving “free” iron [97,98]. Many cells tend to regulate their iron uptake as a mechanism of defense against the formation of the more active hydroxyl radical formed from the Fenton reaction [99]. However, once generated within the cell, the hydroxyl radical is free to attack biomolecules.

3.2.1. Enzymes and coenzymes

Among the most vital substances in the cytoplasm are enzymes. Enzymes catalyze a wide variety of chemical reactions in the cell. No study has investigated the degradation of vital enzymes as part of a photocatalytic inactivation mechanism. Though its effect has not been substantially studied in photochemical disinfection, superoxide is capable of directly inactivating a select group of enzymes [100,101].

However, the initial studies in photocatalytic disinfection pointed to the oxidation of coenzyme A as the mechanism of inactivation [8]. Coenzymes (small non-protein molecules) participate in catalytic reactions as part of an enzyme. Coenzyme A was isolated as a target to be observed largely because the same research group had previously shown that coenzyme A mediates electron transfer between cells and an electrode [102]. It could be possible that attack of coenzyme A plays a role in cell inactivation.

3.2.2. Nucleic acids

DNA is particularly susceptible to oxidative stress. ROS may attack DNA either at the sugar or at the base, giving rise to a large number of products [103]. Attack at the sugar ultimately leads to sugar fragmentation, base loss, and a strand break with a terminal garmented sugar residue. Damage to nucleic acid in photocatalytic systems appears as an indirect result from the generation of superoxide. By using strains of *E. coli*, deficient in genes known to confer resistance to ROS and regulate iron uptake, Gogniat and Dukan [104] suggested that DNA damage resulted from hydroxyl radical attack generated by the Fenton reaction. They observed an increase in susceptibility to photocatalysis by the mutant strains, particularly during recovery. In addition, this may support the observation that DNA can be attacked by at least two different modes, which include direct hydrogen peroxide or superoxide attack and Fenton reaction-generated radicals [104].

4. 4. Disinfection models

4.1. Chick’s Model

Chick’s law expresses the main principles of traditional chemical disinfection [105]. The first-order reaction rate may be expressed as:

$$r = -kN \tag{6}$$

It reduces disinfection to a bimolecular chemical reaction in which microorganisms are treated as molecular species. Chick’s model is a very simplistic formulation, but it has found extensive application where chemical disinfectants such as chlorine, ozone, hydrogen peroxide and chloramines are used [106]. Watts et al. [17] used Chick’s formulation to calculate disinfection rate constants for photocatalytic inactivation of viruses and coliform bacteria from secondary clarifiers in real wastewater effluent.

4.2. Chick–Watson Model

The Chick–Watson model [107] incorporates the concentration–time (*CT*) product concept into Chick’s Law.

Assuming no disinfectant demand (i.e., c and n are constants), then the Chick–Watson model for a batch system is given by:

$$\frac{N}{N_0} = e^{-k't} \tag{7}$$

where k' is the pseudo-kinetic constant,

$$k' = -k[c]^n \tag{8}$$

Rincon et al. [108] demonstrated that the model can sometimes fit observed data for photocatalytic inactivation even though the assumptions may not reflect the underlying mechanisms. Other researchers have resorted to modifications of the models. The main advantage of models based on Chick's law is simplicity. However, it is difficult to make far-reaching conclusions and compare different studies based on the Chick–Watson model. Firstly, Chick's law assumes first-order kinetics and it has been found that disinfection of various microbes (even with chemicals) usually deviates from this assumption [109]. Secondly, parameters, often with no real physical meanings, are introduced to allow the model to fit observed data.

4.3. Delayed Chick–Watson model

The delayed Chick–Watson model [110] is a modification in which a time lag parameter (t_{lag}) is introduced to approximate an initial lag phase in the disinfection process. For $t > t_{lag}$ the pseudo-first-order loss of viability is replicated. The model may be represented as shown below:

$$\frac{N}{N_0} = \begin{cases} 1 & \text{for } t \le t_{lag} \\ e^{-k'(t-t_{lag})} & \text{for } t > t_{lag} \end{cases} \tag{9}$$

The delayed Chick–Watson model has been used by researchers to estimate *CT* values for the photocatalytic inactivation of E. coli (0.8×10^{-5} mg min/l) [69] and *Cryptosporidium* (9.3×10^{-5} mg min/l) [21]. In both of these studies, the hydroxyl radical was shown to be the dominant disinfectant species and the reported *CT* values achieved a 2-log inactivation.

4.4. The Hom model

The Hom model [111] of 1972 presents a generalized differential equation for the time-concentration relationships for the effect of the disinfectant on the microorganism. The expression is given as:

$$\frac{dN}{dt} = -kNt^m c^n \tag{10}$$

In the case where the reaction is zero-order with respect to time and disinfectant concentration, it reduces to the first-order relationship of Chick's Law. However, in the case where $m \neq 0$ and $n \neq 0$ and $c^n\ t = k'$, then the following expressed may be derived:

$$\ln \frac{N}{N_0} = \frac{-Kk't^m}{m} \tag{11}$$

The Hom model is useful for fitting disinfection curves with either an initial lag (when $m > 1$) or trailing curve (when $m < 1$). It cannot replicate both conditions simultaneously.

4.5. Kinetic power law models

One final model is the kinetic power law formulation in which the rate of inactivation is not assumed first-order with respect to microbial concentration. It may be written as:

$$r = -kN^x c^n \tag{12}$$

The integration of Eq. (12) gives the following for the survival ratio of organisms:

$$\ln \frac{N}{N_0} = \frac{-1}{x-1} \ln[1 + (x-1)kc^n t {N_0}^{x-1}] \tag{13}$$

Similar to the Hom model, Eq. (13) can fit observed data displaying shoulders ($x < 1$) or tailing off behavior ($x > 1$).

An example of the kinetic power law model is that used by Wei et al. [112]. In this study they reported a reaction order of $x = 1.06$ for the inactivation of *E. coli* with TiO_2. They also found that the disinfection rate was proportional to the square root of TiO_2 concentration and proportional to incident light intensity within a range of 180–1660 $\mu E/s/m^2$.

5. Mechanistic modeling of photocatalytic disinfection

The main shortcomings of the previous models are: (1) they are applied empirically and; (2) they are not built on the underlying chemical and physical processes of photocatalytic disinfection. Instead, they are built on the assumption of a homogenous chemical reaction, whereas photocatalysis is a heterogeneous process whose overall kinetics varies from those of traditional chemical disinfection and homogenous reactions in general. Therefore, the various constants and fitting parameters developed often have no real meaning in the actual processes being modeled.

The reaction pathways through which TiO_2 is able to inactivate organisms are complex, and mechanism based modeling of photocatalytic inactivation has not been fully explored. Mechanistic models are often preferred over empirical models although they are more cumbersome from a computational perspective. They are expected to be more robust than empirical models and are well suited for application to complex situations that arise in practice, such as modeling changes in radiation source (UV lamps or solar light), addition of other chemicals, and variation in water quality or source.

5.1. Lipid peroxidation mechanism

Given the rising consensus on membrane damage in photocatalysis, a generalized kinetic scheme for lipid peroxidation under photocatalytic conditions may be illustrated as shown below, assuming that the hydroxyl radical is the dominant oxidant in the process.

$$\mathrm{Ti|OH}\bullet + \mathrm{LH} \xrightarrow{R_i} \mathrm{Ti|H_2O} + \mathrm{L}\bullet \tag{14}$$

$$\mathrm{L}\bullet + \mathrm{O_2} \xrightarrow{k_0} \mathrm{LOO}\bullet \tag{15}$$

$$\mathrm{LOO}\bullet + \mathrm{LH} \xrightarrow{k_p} \mathrm{LOOH} + \mathrm{L}\bullet \tag{16}$$

$$2\mathrm{LOO}\bullet \xrightarrow{k_t} \mathrm{products} + \mathrm{O_2} \tag{17}$$

The three principal events in the lipid peroxidation process are initiation, Eq. (14), propagation cycle, Eqs. (15) and (16), and termination, Eq. (17). From this scheme, the kinetics of lipid peroxidation may be developed for photocatalytic systems in which hydroxyl radicals are generated in the extracellular environment. The cells can be treated as compartmentalized micro-reactors within which the radical chain reaction occurs and promotes cell inactivation via the oxidation of lipids (LH), specifically PUFA. Initiation occurs when the hydroxyl radicals on the surface of the catalyst abstracts a hydrogen atom from a membrane fatty acid chain producing a carbon-centered radical, $L^{\bullet}$. The peroxidation of the fatty acids occurs within the cell and it is assumed that the radicals formed during these processes remain in the cell where they are generated.

Although the oxidation of lipids is complex, the predictability of PUFA oxidation kinetics in homogeneous solution [83,84,113]

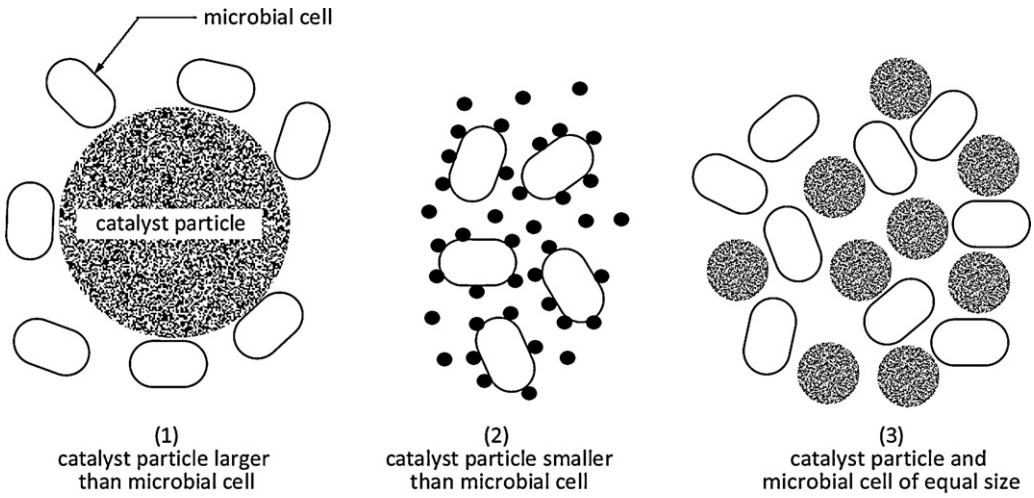

Fig. 4. Relative size interaction of catalyst particle and microbial cell.

and cellular systems [114–118] may allow for the predictable determination of inactivation of microorganisms. The rate of oxygen consumption during the oxidation of the lipids is given as [116,117];

$$-\frac{d[O_2]}{dt} = k_p[LH]\left(\frac{R_i}{2k_t}\right)^{1/2} \quad (18)$$

where [LH] is the oxidizable intracellular lipid concentration, k_p is the propagation rate constant, R_i is the initiation rate, and $2k_t$ is termination rate constant.

The initiation rate is associated with two processes: (1) the generation of radicals; (2) the reaction of the radicals with the membrane lipids. The slower process controls the initiation rate. The generation of radicals is governed by Eqs. (2) and (3), while the reaction of the radicals with lipids is governed by the kinetics of Eq. (19):

$$R_i = k_i[\bullet OH][LH] \quad (19)$$

where k_i is the rate constant of initiation.

As indicated in Eq. (18), lipid peroxidation is often monitored by assessing the rate of oxygen uptake, usually measured with a biological oxygen monitor. However, the products of lipid peroxidation, including MDA, may also be assessed by appropriate techniques, including thiobarbituric acid (TBA) assay and chromatography.

The process of lipid peroxidation should precede the loss of viable cells [119]. In the peroxidation model, inactivation would occur at some threshold of peroxidation (critical level of lipid damage). Even though peroxidation may continue, further damage has no effect on an already inactivated organism. The death rate (disappearance of live organisms) is therefore a function of the lipid peroxidation rate and the critical level of lipids below which organisms die.

The idea of a critical level of damage leading to inactivation is not new. This concept was proposed in the series-event model described by Severin et al. [120]. However, Cronan and Gelmann [89] were able to demonstrate that the reduction of unsaturated fatty acid content below a critical value results in termination of growth and eventual lysis in *E. coli*. The researchers found that the membrane was only functional when at least 15–20% of the total fatty acid content was unsaturated. When the unsaturated fatty acid content dropped below 15%, growth was inhibited and cellular lysis occurred.

The question regarding the precise mathematical relationship between the rate of lipid peroxidation and the rate of cell inactivation is still unanswered. In the few studies that confirm lipid peroxidation during photocatalysis [9,20,81,91,121], there was no quantitative relationship established between death kinetics and peroxidation. Therefore, still to be determined is the relation between photocatalytically induced lipid peroxidation and cell death in terms of time and concentration dependency. However, the general trend observed is that the products of peroxidation increase as cells are inactivated.

5.2. Microbe–catalyst interaction model

The development of a mechanistic model requires an understanding of the hydrodynamic interactions between the catalyst and the microorganism. From a particle size perspective, the interaction may be viewed in three ways as shown in Fig. 4; (1) the catalyst particles are larger than the microbial cells. This may be the case when catalyst particles agglomerate forming relatively large aggregates or when the catalyst forms a continuous immobilized surface (e.g., thin film applications); (2) the catalyst particles are much smaller than the microbial cells as is the case for ultra-fine catalysts in suspension; (3) the catalyst particles and the microbial cells are of equal size and mutually interact.

5.2.1. Adsorption model approach

The kinetics of a photocatalytic process may be appropriately described by an adsorption-type model such as Langmuir or Freundlich under conditions described in Fig. 4(1) and (2). In the first case, a number of microbial cells can become attached to a single catalyst particle or a continuous surface. Secondly, when the catalyst is much smaller than the cell, several catalyst particles can adhere to a single microbe. However, a major challenge in the application of adsorption models to disinfection is the difficulty in resolving microbe–catalyst interactions, particularly given their relative sizes and bacterial complexity.

In classical heterogeneous catalysis there are at least five fundamental steps which occur:

(1) The transfer of aqueous reactants to the solid surface.
(2) The adsorption of the reactants on the surface.
(3) The reaction between adsorbed species.
(4) The desorption of products.
(5) The transfer of products from the solid surface (or inter-phase) to the bulk liquid phase.

In order for the disinfection reaction to occur, microbes, like molecular reactants, are required to be in contact with or in very close proximity to the photocatalyst surface. However, while adsorption of molecules to substrates has been well defined, the process differs significantly for microorganisms [122,123]. Firstly, so-called microbial adsorption may be more appropriately called adhesion [124]. Adsorption usually involves particles of molecular or colloidal scales, whereas microbes are of a much larger scale, and they are often as large as, or larger than the catalyst particles. It follows that any agreement with the classical isotherms will depend on the relative size of the microorganism and the catalyst particle. Size can affect the mechanism of sorption, rates of transport, and equilibrium capacities [124].

Secondly, a bacterial surface is much more complex than soluble molecules or inanimate colloids. The surface may be composed of different functional groups such as carboxylic, phosphodiester, phosphoric, amines, and hydroxyl moieties [125,126]. Under most physiological conditions, there is a net negative charge which arises from the ionization of proton-active functional groups attached to the cell wall polymers. Some of these groups may associate or dissociate protons upon changes in pH or ionic strength of the suspending fluid, but also upon approach of a charged surface.

For a comprehensive review of the sorptive behavior of microorganisms to solid surfaces, the reader is referred to the work of Bitton and Marshall [124] and Poortinga [127]. Bitton and Marshall [124], for example, eruditely discuss the application of diffusion-controlled processes, thermodynamic analysis and rate equations for bacterial sorption. The application of the Langmuir isotherm for suspended microbial cells is described here to illustrate the development of a suitable model for photocatalysis.

A number of authors found that the adsorption of viruses [128,129] and bacteria [130,131] followed the Langmuir isotherm. No study has been specifically performed to determine the sorption kinetics of microbes to photocatalyst particles. Therefore, the principal findings of related studies are applied here.

A Langmuir equilibrium constant (K) for adsorption can be expressed by:

$$K = \frac{q}{(Z-q)N_e} \tag{20}$$

where N_e is the number of cells in aqueous suspension at equilibrium (cell/ml), q is the number of cells adsorbed per unit mass of catalyst particles at equilibrium (cells/mg) and Z is the adsorbent capacity, that is, number of sites per unit mass of catalyst particles (sites/mg).

From the equation, it is clear to see that q is proportional to the surface coverage of cells and $(Z-q)$ is the number of vacant sites. Therefore, it is possible to define another equilibrium constant, b, in terms of surface coverage fraction, θ and equilibrium bacterial concentration:

$$b = \frac{\theta}{(1-\theta)N_e} \tag{21}$$

By rearranging Eq. (21), the following traditional expression for the Langmuir surface coverage is achieved.

$$\theta = \frac{bN_e}{1+bN_e} \tag{22}$$

In photocatalytic studies, adsorption models have traditionally been applied for the removal of chemical pollutants [56,64,132–134] and not microbes. However, Marugán et al. [10] developed their disinfection model for TiO_2 based on Langmuir–Hinshelwood-type kinetics. Assuming a simplified inactivation mechanism in which "undamaged" cells become "damaged" and eventually progress to an "inactivated" state, they propose the following expressions:

$$\frac{dN_{\text{undam}}}{dt} = -k\frac{KN^n_{\text{undam}}}{1+KN^n_{\text{undam}}+KN^n_{\text{dam}}} \tag{23}$$

$$\frac{dN_{\text{dam}}}{dt} = k\frac{KN^n_{\text{undam}}-KN^n_{\text{dam}}}{1+KN^n_{\text{undam}}+KN^n_{\text{dam}}} \tag{24}$$

Three model parameters are defined: the kinetic constant, k, a pseudo-adsorption constant, K (which accounts for interaction between the catalyst and the microbes), and an inhibition parameter, n (which accounts for increasing concentrations of disinfection products). N_{undam} and N_{dam} are the counts of "undamaged" and "damaged" bacteria respectively, the sum of which gives the total surviving population of bacteria.

A major benefit of this model is that it is flexible for the description of photochemical inactivation kinetics. The model parameters have real significance in relation to the underlying processes. The inhibition parameter (n), for example, accounts for the slower rate towards the end of the process as products accumulate. It also represents the reaction order with respect to bacterial concentration as the value x does in the kinetic power law model. Like Wei et al. [112], Marugán et al. [10] also found n to be greater than 1. The other constants, k and K, were shown to have discernible dependence on catalyst concentration.

However, due to the complexity of the underlying processes which occur once a microbe is in contact with the catalyst, the authors were careful in calling their model a Langmuir–Hinshelwood "type" kinetic model. Therefore, the inactivation process is treated as two simple unimolecular elementary reactions. The model does not provide a specific fundamental process or set of steps by which the inactivation process occurs. It falls short of incorporating any biological information about the organisms being inactivated. This means that it would be difficult, without much prior experimentation, to accurately predict the disinfection rates for different organisms or the same organism grown under different conditions. Nonetheless, this may be solved by determining the values of k, K and n for different organisms or a collection of organisms.

However, if lipid peroxidation is assumed to be the dominant inactivation process, the following expression may be derived:

$$R_i = k_i\theta_{\bullet OH}\theta_{LH} \tag{25}$$

where $\theta_{\bullet OH}$ and θ_{LH} are the surface coverage of radicals and lipids. By applying the Langmuir model, the rate of initiation is given as:

$$R_i = k_i\frac{b_{LH}[LH]b_{\bullet OH}[\bullet OH]}{(1+b_{LH}[LH]+b_{\bullet OH}[\bullet OH])^2} \tag{26}$$

In Eq. (26), b is the adsorption equilibrium constant for the respective subscripts. This expression may be further be simplified to Eq. (27) for the case where $\theta_{LH} \ll 1 \ll \theta_{\bullet OH}$.

$$R_i = k_i\frac{b_{LH}[LH]}{b_{\bullet OH}[\bullet OH]} \tag{27}$$

Therefore, the rate of oxygen uptake for one cell is given as:

$$-\frac{d[O_2]}{dt} = k_p[LH]^{3/2}\left[\frac{k'}{2k_t[\bullet OH]}\right]^{1/2} \tag{28}$$

where $k' = k_i(b_{LH}/b_{\bullet OH})$.

Although these relationships are purely theoretical, they do demonstrate that the development of a mechanistic model based on the oxidation of lipids is plausible given the existing knowledge on lipid oxidation in cellular systems. The actual dependence of peroxidation rate on the various parameters will be determined from experimentation. In addition, the term $(k_p/2k_t)^{1/2}$ is generally taken to be the oxidizability or susceptibility to peroxidation in autooxidation studies of lipids [116–118].

5.2.2. Collision model approach

In a system defined by a suspension of solid catalyst and microorganisms, particularly when the particles are of similar size, a collision model may better describe the process of interaction between the particles and the means by which the photocatalytic reaction occurs. In classical collision theory, reactants must collide with sufficient energy and with the right alignment for a reaction to occur. The same can be said of the microbe and catalyst, except that the collision results in the initiation of the lipid peroxidation process. Therefore, collision is a prerequisite for disinfection, but does not instantaneously result in microbial inactivation.

The rate of initiation can be expressed as:

$$R_i = k_i N m_{\text{cat}} \tag{29}$$

where N is the concentration of bacteria, m_{cat} is the concentration of catalyst, and k_i is the initiation rate constant.

Catalyst particles which have been photosensitized can be considered "activated" particles. Only activated particles carry the hydroxyl radical capable of initiation the reaction. The interaction with activated particles must be maximized to increase the reaction rate. In a suspension of catalyst particles, much of the light tends to be absorbed by the particles closer to the light source. Therefore, in any given instance, there is always a fraction of the suspension which is exposed to light and a portion that is blocked, resulting in a distribution of activated particles [135].

At high catalyst concentrations the ratio of activated catalyst particles to "non-activated" particles is low due to the high attenuation of light in concentrated solutions. The chances of an organism colliding with non-activated particles are much higher than with activated catalysts. At lower catalyst concentrations the reverse is true. However, at very low concentrations the high ratio of activated catalyst particles cannot compensate for the sharp reduction in collision frequency. Therefore, an optimal concentration can be defined at which collision frequency with activated particles is maximized. Most authors report that above concentrations of 1 g/L of catalyst there is no significant increase in disinfection rate [10–12,26,69,92,136].

The reaction rate constant for initiation is therefore the product of the total number of collisions, the fraction of activated catalyst, and an efficiency factor that accounts for the fact that not each of these collisions will result in initiation.

$$k_i = \sigma \times f_{\text{cat}}^{\text{act}} \times e \tag{30}$$

where σ is the total number of collisions, $f_{\text{cat}}^{\text{act}}$ is the fraction of activated catalyst, and e is the efficiency factor.

The fraction of activated catalyst is a function of light intensity, catalyst concentration, particle size, optical properties, and reactor configuration. It is directly related to the ratio of the amount of energy absorbed by the suspension to the maximum theoretical absorption capacity of the entire suspension [94,135].

In the collision model, the kinetic law will mainly be governed by the rate of initiation and the microbial concentration. The rate of oxygen uptake for one cell is given as:

$$-\frac{d[\text{O}_2]}{dt} = k_p[\text{LH}]N^{1/2}m_{\text{cat}}^{1/2}\left(\frac{k_i}{2k_t}\right)^{1/2} \tag{31}$$

The collision model predicts that the overall rate should be proportional to the square root of catalyst concentration. Excessive catalyst concentration would be impractical and has proven to be ineffective in achieving further increases in the reaction rate [10–12,26].

5.3. Series-event model

A series-event model was proposed by Severin et al. [120] for the disinfection by UV irradiation and later for chemical disinfection [137]. The model assumes that a series of discrete damage levels exist and that each level or event is a unit of damage that occurs in a stepwise manner until the organism reaches a threshold level of damage. Organisms are assumed to survive if the damage is below the threshold. The model can be represented as follows:

$$D_0 \xrightarrow{k_1} D_1 \xrightarrow{k_2} D_2 \ldots \xrightarrow{k_i} D_i \ldots D_{n-1} \xrightarrow{k_n} D_n$$

Each step is characterized by first-order kinetics with respect to a constant concentration of chemical disinfectant. Each damage level D_i has a kinetic constant k_i and n is the threshold level of damage. The concentration of the disinfectant is assumed constant, so that k_i is really a pseudo-kinetic constant which can be represented by kC. The disappearance of organisms at damage level D_0 is given as $-k_1N_0$ and for level D_1 the expression is k_1N_0–k_2N_1, where N_0 and N_1 are the concentrations of the organisms at the two damage levels respectively. The total number of surviving organisms is therefore the summation of all organisms below the threshold damage level, i.e., up to D_{n-1}.

The main limitations of this model are: (1) it requires a large number of damage levels to accurately describe inactivation and (2) it is not flexible for analyzing disinfection data since it can only be used to analyze concave curves. In addition, the model is very generic because it does not make any assumptions about the disinfection mechanism [137]. By assuming that the kinetic constant is the same at each level, the following generalized expression can be derived for the series-event model.

$$\log\frac{N}{N_0} = -kt + \ln\left(1 + \sum_{i=1}^{n}\frac{(kt)^i}{i!}\right) \tag{32}$$

5.4. Multi-target model

The multi-target model is similar to the series-event model, in that it assumes that each organism contains a finite number of discrete critical targets (n_c), each of which must be attacked for full inactivation of the organism. When derived for batch reactor conditions, the multi-target model takes the following form.

$$\frac{N_s}{N_0} = 1 - (1 - e^{-kt})^{n_c} \tag{33}$$

All the targets are assumed to be equivalent and the damage is randomly distributed among the targets. As a target is destroyed, the probability of hitting the remaining targets is reduced. The expression $(1 - e^{-kt})$ is the probability of inactivating a specific target.

6. Conclusions

It is obvious that accurate kinetic information is a prerequisite for an effective design of a photocatalytic disinfection system. Inaccurate estimation of a system's performance may lead to inadequate disinfection and wasted financial investment. Therefore, a robust mechanistic model is needed to determine the most efficient combination of contact time, catalyst dose and light exposure.

Review of the available literature clearly shows that a robust mechanistic model for photocatalysis is still quite away from being developed. Even some of the recent proposed mechanistic models are probably better described as semi-mechanistic or semi-empirical. Even though they are able to fit observed data through difficult computation, they really do not resolve any of the complexity underlying the inactivation process. Incorporating biological information or parameters into the model may be useful in differentiating the susceptibility of different organisms. Moreover, there

is a wide pool of biological information available on microorganisms that may prove useful in the development of a mechanistic model.

Understanding the fundamental reactions which occur during disinfection will be important in improving and modeling the process. To this end, it is very apparent that lipid peroxidation will play a very important role in the development of a mechanistic model, at least for bacteria and possibly viruses. Research on photocatalytically induced membrane peroxidation is still relatively new. To date, no specific study has explored the kinetics of peroxidation for cells exposed to a source of radicals from photocatalysis. Nevertheless, the literature on lipid peroxidation, initiated by other radical sources, is extensive and provides a sound basis for guiding photocatalytic disinfection research.

However, one of the most important questions regarding how the kinetics of lipid peroxidation is related to death kinetics is essentially missing from the body of literature. Still to be determined is the relation between photocatalytically induced lipid peroxidation and cell death in terms of time and concentration dependency. Future work in this area will need to quantitatively relate lipid peroxidation and photocatalytic susceptibility to the decline in cell viability, the parameter usually measured in such studies. All of these observations point to the importance of fundamental research in filling the existing knowledge gaps, which will allow photocatalysis to become even more competitive with traditional disinfection techniques.

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